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9:15

MECHANISM OF TILT TABLE INDUCED HYPOTENSION AND BRADYCARDIA IN PATIENTS WITH NEURALLY MEDIATED SYNCOPE

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It is postulated that neurally mediated syncope (S) is triggered by the stimulation of ventricular mechanoreceptors resulting from changes in ventricular volume and or contractility. To further elucidate this mechanism, we evaluated changes in left ventricular end diastolic volume(EDV), end systolic volume (ESV), and ejection fraction (EF) by 2 dimensional echocardiography (2DE) during head-up tilt (HUT) in 17 pts with recurrent S. Nine pts had vasovagal or vasodepressor S induced by HUT (+HUT) and 8 pts had no S during HUT (-HUT). There were no significant differences in the baseline EDV, ESV, and EF in pts with +HUT compared to those with -HUT. The percent change (%Δ) in ventricular volumes at 10 minutes of HUT compared to baseline, and the EF at 10 minutes of HUT (EF 10) are displayed below:

	N	%ΔEDV	%ΔESV	EF 10
+HUT	9	-32 ± 22	-41 ± 25	67 ± 10
-HUT	8	-12 ± 21	-12 ± 30	55 ± 15
p value		.07	<.05	<.01

Conclusion: The mechanism of syncope in +HUT pts appears to involve an exaggerated decrease in ventricular volumes and an elevated EF when compared to (-)HUT pts. These data support the theory that stimulation of ventricular mechanoreceptors initiates neurally mediated S.

9:30

SEQUENTIAL CATECHOLAMINE CHANGES DURING UPRIGHT TILT: POSSIBLE HORMONAL MECHANISMS RESPONSIBLE FOR PATHOGENESIS OF NEUROCARDIOGENIC SYNCOPE

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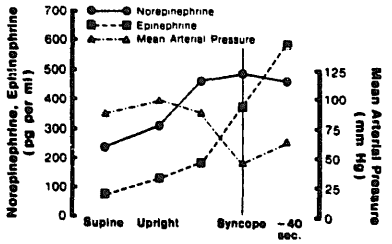
Norepinephrine (NE) and Epinephrine (EP) levels were compared at baseline, initial upright tilt, every 90 seconds during 70° head up tilt (HUT), at initiation of syncope or termination (15 minutes in normals) and 40 seconds after termination in 10 pts. with neuro-cardiogenic syncope (NCS) and 5 normals. Baseline EP was higher in pts. with NCS vs normals, 76±28 vs 27±5 (P < 0.003). NE increased in both groups as HUT progressed. However, in pts. with NCS during HUT induced syncope NE failed to rise despite marked fall in mean arterial pressure, while EP rose from baseline of 76±28 to 383±311 (P < 0.0001) (figure). Rise in EP in normals was insignificant, 27±5 to 58±33 (P=NS).



Conclusion: Combination of sympathetic inhibition and B-2 adrenergic stimulation is the possible mechanism of HUT induced hypotension and syncope in pts. with NCS.

9:45

VASOVAGAL REACTIONS MAY OCCUR BEFORE AND AFTER RE-INNERVATION IN ORTHOTOPIC CARDIAC TRANSPLANTATION

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Stimulation of left ventricular receptors is believed to be responsible for vasovagal reactions. Orthotopic cardiac transplantation (OCT) may result in permanent denervation of the donor heart so that vasovagal reactions would not be expected. 10 patients with OCT underwent tests for parasympathetic innervation of the donor heart before and after atropine infusion, (0.02mg/kg), and on a second occasion had prolonged orthostatic head-up tilt with a saddle support. Twelve non-syncopal controls were also tilted. Patients were 34 (±15) months post-operation. Native (NHR) and donor (DHR) sinus rates were monitored using an oesophageal pill-electrode during vagal testing and throughout tilting.
Testing suggested that 3 patients 40(±13) months from operation had evidence of vagal re-innervation, (Group I). 7 others (30(±15) months), had no such evidence, (Group II). During tilt all patients in Group I had vasovagal reactions, and there was a mean fall in NHR of 25±3bpm, in DHR of 36±34 bpm, and in mean arterial blood pressure of 60±9mmHg, a the time of the vasovagal reaction. In Group II there were 4 tilt-induced vasovagal reactions, NHR fell by 32±13bpm, DHR did not change, and MABP fell by 47±5mmHg. Vasovagal reactions occurred in 7 out of 12 controls (58%).

CONCLUSIONS: These data suggest that vagal re-innervation may occur after human OCT, causing donor heart bradycardia in some cases during the vasovagal reaction. It has not been demonstrated, however, that afferent re-innervation has occurred, and the absence of donor heart bradycardia during vasovagal reactions in Group II suggest that intact left ventricular receptors may not be necessary for tilt-induced vasovagal reactions.

Wednesday, March 6, 1991
8:30AM–10:00AM, Room 360, West Concourse
Advances in Intravascular Ultrasound I

8:30

FLOW-DIRECTED, BALLOON-FLOATATION INTRAVASCULAR ULTRASOUND CATHETER FOR PERCUTANEOUS PULMONARY ARTERY IMAGING AND INTRACARDIAC ECHOCARDIOGRAPHY

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The feasibility of imaging pulmonary arteries and visualizing cardiac structures using wire-guided intravascular ultrasound catheters under fluoroscopic control has been demonstrated in animals and humans. To assess whether such imaging could be performed without the hazard of fluoroscopic radiation and without the requirement of guide-wires, we developed a 4.8 F, 20 MHz ultrasound catheter (length 110 cm) with an inflatable balloon at its tip and employed it percutaneously in 5 dogs on 10 occasions. Introduced through a sheath in the jugular vein/femoral vein, continuous IVUS imaging displayed the passage of the catheter through the SVC into the RA. Images of the RA chamber, its free wall and the atrial septum could be seen. With the catheter tip in RA, the balloon was inflated. The blood flow then carried the catheter through the RV into the main pulmonary artery and then into the distal PA branches, in a manner similar to conventional Swan-Ganz catheter passage, without the need for fluoroscopy, undue manual manipulation or guide-wires. Either sterile water or air injected into the balloon supported its intracardiac travel with equal efficiency. Once the catheter tip reached the distal PA, the balloon was deflated. Imaging could be performed both with deflated balloon and with water-filled balloon. Dynamic, high resolution cross-sectional images of the distal, middle level and proximal pulmonary arteries could be obtained with ease. During gradual withdrawl of the catheter, images of pulmonary arteries, pulmonic valve, RV chamber, tricuspid valve and RA chamber could be visualized. There were no catheter knots, ventricular arrhythmias or other complications. We conclude that intracardiac imaging of the right heart structures and intravascular visualization of the distal and proximal pulmonary arteries can be performed percutaneously with safety and ease using balloon-tipped flow directed intravascular ultrasound catheters and that this mode of imaging could have considerable clinical applications.